into optic and spinal types. As to suspension, he had found it unsatisfactory, and had been unfavorably impressed with it. It had seemed to him better in spastic cases, such as myelitis.

Dr. WAITZFELDER had been using suspension in a case of spastic paralegia, and it had made that case worse, as well as several other cases of myelitis; but he had noted considerable improvement in gait in several patients with locomotor ataxia who had been subjected to this method of treatment.

Dr. LESZYNSKY had observed a few favorable results in the employment of suspension in spinal cases. He had also used it in a case of paralysis-agitans three or four times with good effect.

Dr. GRAY then closed the discussion. He said that Fournier's and Rumpf's specific cases showed great improvement under treatment, but this had not been his own experience. He believed there were certain cases where there would be great difficulty in distinguishing peripheral from central symptoms. As to the matter of self-limitations mentioned by Dr. Starr, he saw no analogy between tabes and phthisis. In treatment he preferred to follow French authors and employ inunction in specific cases rather than potassic iodide, for the results were much better.

FREDERICK PETERSON, M. D.,
Secretary.

AMERICAN NEUROLOGICAL ASSOCIATION TRANSACTIONS.

DR. B. SACHS, of New York, read a paper entitled

POLIOENCEPHALITIS SUPERIOR (NUCLEAR OPTHALMOPLEGIA) AND POLIOMYELITIS.

It is a rare experience in neurological matters to have the pathology of a disease unravelled as quickly as was done in the case of those clinical groups of symptoms which we know as opthalmoplegia externa and interna.

The paralysis of the muscular apparatus of the eye was soon discovered to be due, in most cases, to a lesion or lesions affecting the nuclei of the nerves which govern the various ocular muscles. In this nuclear paralysis, the nuclei of the oculo-motor nerves play a most important *role*, though the nuclei of the fourth and sixth nerves are involved frequently enough.

The relation of the bulbar process to poliomyelitis was firmly proved by cases of bulbar paralysis which were associated with symptoms resembling those of a progressive muscular atrophy or a chronic anterior poliomyelitis, and furthermore by cases of typical progressive muscular atrophy which, in their terminal stages, developed bulbar symptoms.

On the strength of this clinical analogy, Hutchinson, Mauther, and Birdsall were struck with the pathological resemblances between the diseases affecting the ocular and spinal nuclei; and Wernicke proposed to call the affection of the oculo-motor nuclei a polioencephalitis superior, whence it followed that the bulbar paralysis might well be styled polioencephalitis inferior. While the analogy with poliomyelitis had been proven beyond the shadow of doubt for the bulbar cases, Wernicke's theory needed further proof as regards the cases of total opthalmoplegia externa and interna. Heretofore but one case has been recorded (by Seeligmüller) in which the symptoms of a chronic poliomyelitis were associated with those of polioencephalitis superior, and with the exception of the cases of Henoch and Buzzard, in which an ocular (nuclear) paralysis occurred in the course of an acute poliomyelitis anterior, I know of no cases which exhibit this interesting association of symptoms.

The history of this case is as follows:

H. M., aged forty, is a man of robust build, unusually intelligent, and one who has experieneed all the vicissitudes of life. He was born in this city, and has been married fourteen years. Has one brother living and healthy, and one brother who died from want of water, as the patient says, on the desert of Arizona. Both parents are dead; the

father died of yellow fever in New Orleans in 1858; the mother died of a paralysis which lasted seven or eight years and began by turning-in of both feet. The paralysis of the legs became complete; she died at the age of seventy-four. The patient went to school at Syracuse, N. Y. As a boy he had frequent "bilious" attacks associated with headaches and vomiting, was otherwise in robust health. He attended school until the age of twelve; went to New Mexico at the age of fourteen, and there learned a trade.

When sixteen years of age, one day after reading several hours got up to stretch himself, but fell back unconscious against a hot stove, burning the left temple severely —the scar is visible at the present day. He was insensible for several hours and was then put to bed; knew nothing of what had happened until he saw doctors around; no paralysis followed. A second similar attack, again after reading, occurred three months later, from which he quickly recovered. A third attack occurred, but the date of this he cannot recall. No further sickness until the age of twenty, when he was in Peru and was steward on a United States steamer. While on shore he fell from a horse, striking the right elbow and injuring the arm. Recovered completely from this fall.

At the age of twenty-five was in Europe; felt one day a severe pain in the left eye; engaged passage at once for New York; reaching there a few weeks later. By that time the left eye was closed. Went to sea again (to Australia on a sailing vessel), and during this trip noticed that the right lid was also beginning to droop. He was treated in Melbourne by electricity, but the condition remained stationary for several months. After that a slight improvement is said to have set in the left eye, but the right eye grew worse; both pupils were dilated (physician's statement). Had double vision all the time, and small ulcers formed on the left eye. The Australian physicians suspected tumor of the brain. He had severe headaches at the time; the left eye became inflamed and was in such a bad condition that the physician advised enucleation, but the patient objected. After a short trip at sea the inflammatory condition was at an end.

It was about this time that he began to be suspicious of his legs; for one day, while walking on deck, his right knee gave way. A few days later the same accident occurred. Nevertheless, he joined a ship from Australia to California. When thirty days out he had to refuse duty as steward, as he could not move his right thigh, leg or toe a single inch. The captain ordered hot steam bath and gave him blue pill and black draught. He went to Oregon next —now thirteen years ago. He remembers that when there he could not hold water nor contain feces. His left leg was not affected at any time. Iodide treatment was proposed. Patient objected, on the ground that he had never had any syphilitic affection. In consequence of the paralysis of the right leg he was compelled to go about on crutches. The doctor who examined him found a sensitive point between the shoulder-blades, and ordered blisters, and strychnine internally. In six months' time he was able to walk with the assistance of a stick. He could use the hip and knee-joint, but could not move ankle or toes. The eyes remained in about the same condition; he was not worried by them. He undertook contract work on a railroad in Panama, and there contracted severe malaria.

One year and a half ago he was stricken down with prolonged fever. As soon as he recovered from this he traveled about considerably; finally went to Jacksonville, where he got a thorough drenching, which was followed by severe chill. One day he tried to read the papers but could not see anything. Last year, on his return to New York, he was examined by Dr. E. Grüning, who performed an iredectomy of the right eye, which did not improve vision. Later on Dr. Grüning raised the left eyelid by operation and restored vision to that eye. Went to Panama in May, 1888, and returned to New York about seven weeks ago.

For the past four weeks he has been an inmate of the Montefiore Home for Chronic Invalids, where I have had the opportunity of studying the case. He denies ever having had gonorrhœa or syphilis, and an examination of his body reveals no symptoms of the latter. He has been

moderate in sexual matters and has never been a hard drinker. Has smoked innumerable cigarettes for years. No loss of consciousness has occurred since the attacks recorded above. Does not suffer from headaches, and but for the condition of his eyes and of his right leg would feel entirely well.

Present Condition.—Strong, well-built man; heart sounds normal; no enlargement of liver; slight enlargement of spleen; other thoracic, and abdominal organs normal. The most striking feature of the patient's appearance is the double ptosis, at present more marked on the right than on the left, in consequence of the operation on the left eyelid. Slight lateral nystagmus of right eye. In this eye, also, maculæ cornæ, old iritis with exclusion of pupil dilated. A transparent, thin membrane has grown upward, covering nearly one-half of left pupil. The results of my examination of the eyes, which were kindly corroborated by Dr. E. Fridenberg, are as follows:

O. D.—Paretic—rectus internus, rectus externus, and inferior oblique. Paralyzed—levator palpebrarum, rectus superior, obliquus superior, and ractus inferior. Associated movements with the left eye do not differ from those attempted singly.

O. S.—Paretic—rectus externus, rectus internus, and superior oblique. Paralyzed—levator palpebrarum, rectus superior, rectus inferior, and obliquus inferior. Paralysis of iris (light reflex abolished), ciliary muscle normal. Accommodation reflex good. Media apparently clear in both eyes. Vision, left eye, $\frac{20}{c}$. Right eye, = 0.

Ophthalmoscopic Examination.—Left papilla normal; right papilla cannot be examined.

No change in the facial distribution. Hearing normal on right side; on left side somewhat diminished, but normal bone conduction. Tongue protruded straight, slight fibrillary movements. Sensation of face and tongue normal in every particular. Smell and taste normal on both sides. The left arm appears to be slighly larger than the right, but grasp is equally strong on both sides. Sensation normal to touch and pain. Distinguishes numbers written on

arm with great ease. No reflexes to be obtained in upper extremities. No difficulty in respiratory or abdominal muscles.

Lower Extremities.—Marked atrophy of right leg from hip downward. Largest circumference on right side; hip, 14¼ inches; left side, 19½ inches; right calf, 10 inches; left calf, 12½ inches. Patient can flex knee very little, but cannot move toes of the right foot. Walks by exclusive use of posterior thigh and leg muscles. Muscular excitability lost in right thigh. No disturbance in sensation except apparent diminution of pain sense on inner aspect of right thigh. The left thigh and leg muscles show normal myotatic excitability and absolutely normal sensation. No change to be noted in any respect in leg of left side. There is no ataxia of either leg and none in the upper extremities. No Romberg symptoms. The knee-jerks are lost on both sides and cannot be elicited by Jendrássik's method. All cutaneous reflexes sluggish but present.

The electrical examination reveals no change in any of the muscles of the face, of the upper extremities, or of the trunk, nor in the left leg; but marked degeneration reaction exists in the anterior thigh and leg muscles. The vasti and anterior tibial muscles are atrophied to such an extreme degree that no action could be obtained with currents at command.

This history can be summarized in a few words: A man in perfect health, without any specific alcoholic or hereditary taint, is affected with a slowly-developing paresis or paralysis of all of the ocular muscles. This condition is scarcely fully established before a weakness of the right leg is noticed by giving way of the knee. This weakness is developed into a most marked paralysis, associated with extreme atrophy. The symptoms remain restricted to the right leg, become retrogressive, and have not to this day effected the opposite leg. The arms remain entirely normal. The transitory bladder and rectal symptoms were probably due to an extension of the inflammation of the gray matter, and do not imply, to my mind, the existence

of a transverse myelitis, acute, subacute, or chronic. No other interpretation can be put upon these symptoms, except to say that in the course of a chronic nuclear paralysis of the eye a subacute poliomyelitis set in. Both in the eyes and in the leg the disease developed in the same fashion, and has practically remained stationary for years.

It will hardly be necessary in this paper to prove the diagnosis of subacute poliomyelitis in this case, and, considering the rarity of poliomyelitis in the adult, it would be strange, indeed, if the occurrence of such an affection in the course of a polioencephalitis superior were a mere coincidence. It seems to me to prove positively that the ganglion cells of the anterior horns of the spinal cord are subject to the same pathological changes as the large nuclear cells on the floor of the third and fourth ventricles.

The involvement of the iris in the one eye (the condition of the other could not be examined) takes his case out of the category of cases of ophthalmoplegia externa. According to most authors, an opthalmoplegia externa, with paralysis of the iris, would compel one to refer the lesion to the base of the brain; but since Westphal and Spitzka have plausibly shown that the nuclei for the accommodation and light reflex lie anteriorly and away from the remaining oculo-motor nuclei, it is readily seen that these nuclei, also, one or both, may be effected by the extension of the inflammatory process. It is in this way that I explain the affection of the iris in this case. Since the accommodation reflex remained normal, it is natural to infer that the ciliary and iris nuclei must be some appreciable distance apart. We must be careful, however, not to be too positive in such assertions, for Thomsen has recorded cases in which there were distinct paralysis of various ocular muscles with only the slightest involvement of a few of the nuclear cells, and, strangest of all, one case of paralysis of associated vision upward, due to a gummatous infiltration of the *oculo-motor root fibres*, whereas the nuclei were found entirely normal. It is for this reason, also, that I believe that the determination of the exact location of the various subdivisions of the oculo-motor nucleus, on clinical grounds

only, has been carried too far. This question can be settled in no other way but by the experimental method, or by noting to what extent clinical and post-mortem records tally.

One other point in the case demands explanation: The knee-jerk is absent on both sides. The first suspicion was that of an accompanying tabes dorsalis, as in Westphal's well-known case; but this supposition must be abandoned, since a close examination with this end in view has shown the absence of every other important symptom of tabes. The absent reflex on the left side must, therefore, be regarded as the only evidence of the extension of the process in the spinal cord to the left half of the cord; but, at the same time, the normal condition of the muscles, the normal electrical reactions, and the total absence of atrophy prove that that side can be effected but very little.

The chief value of my case is, that it proves the close relationship between the gray matter at the floor of the third and fourth ventricles and the anterior gray horns of the spinal cord.

Wernicke chose the term polioencephalitis superior wisely enough; but Strümpell's polioencephalitis, a supposed cortical disease, has caused some confusion. Strümpell's theory and disease lack proof, and for the present we need not decide whether we shall have to add a polioencephalitis suprema to polioencephalitis superior.

Dr. SPITZKA said the report of the case had been so complete that, as there was but one other such case on record, it did not admit of either criticism or comparison. He referred to Thomsen's case of unilateral nuclear paralysis, where there had been gummatous infiltration on both sides, the explanation of which was to him quite impossible.

Dr. SACHS asked if fibres could be traced up to the ciliary nuclei.

Dr. SPITZKA answered in the affirmative, and made blackboard drawings illustrative of their course.

HYPEROSTOSIS CRANII.—Dr. W. N. BULLARD, of Boston, then presented for Dr. J. J. PUTNAM a skull which was a remarkable example of this condition. The case had been

reported to the association two years before. The patient was a woman, thirty-one years old at death. The chief symptoms had been headache, broadening of the head, dropping out of the teeth, loss of hearing, and vertigo, beginning gradually some years ago. There had been extreme exophthalmia. There had been no retinal changes. Extensive pachymeningitis had been discovered at the autopsy. There were thinning and atrophy in parts of the skull. The orbital cavities were greatly diminished in size. Virchow considered hyperostosis cranii due to inflammatory changes. In this case probably the inflammation had originated at the ear. Dr. Putnam had a patient now with similar symptoms, in whom the exostosis were first noticed in boyhood, and desired the opinion of members as to the justifiability of removal of certain exostoses for the relief of pain. Dr. Bullard himself thought it might be difficult to determine which of the exostoses produced the pain. Some of the exostoses were very diffuse, and the operation might have to be extensive.

The PRESIDENT believed that the pain would be more apt to originate from basal regions, possibly dural inflammation about the issuing nerves. The jaw in this case was interesting because of its senile conformation and angle, despite the patient's youth.

SPONTANEOUS DEGENERATIVE NEURITIS OF THE BRACHIAL PLEXUS.—Dr. W. M. LESZYNSKY, of New York, read a paper with this title. The patient was a laborer, aged thirty-eight. He had first had pain in the left shoulder, shooting down the arm, which had been ascribed to exposure to wet. There had been then no involvment of the shoulder-joint and the motility of the arm had been unimpaired. All the muscles had reacted well to faradaism, except the deltoid, which had been atrophied. There had been no sensory disturbance, but there had been pain on pressure. Gradually other muscles had become paralyzed, until a large number of the arm-muscles had been useless. The paralysis had been accompanied by pain, so excruciating that the patient could not sleep at night. A feeling of numbness had extended from the shoulder down the arm

over the radial distribution, and a gradual anæsthesia analgesia had supervened throughout the same area. The faradaic excitability had disappeared, and there had been galvanic superexcitability. Then he had begun gradually to improve, and would ultimately recover. The case had been remarkable in its severity, in its idiopathic origin, and in the escape of the median and ulnar nerves from the inflammatory process. Not more than one case, had to his knowledge been found in literature.

Dr. PRINCE thought it would be difficult in the early stages of such a case to distinguish it from progressive muscular atrophy of the shoulder type. He recalled a case of his own in which there had been every reason for considering it to be neuritis. It had begun with cramps, such as were observed in writer's cramp, and it had been several years before other symptoms proved the case to be one of progressive muscular atrophy. The most common cause in such cases as the author's was traumatic arthritis, but generally the results were slight.

Dr. W. R. BIRDSALL, of New York, had seen this case before, and was impressed with the idea that it might be a periarthritic affection, but the author's careful study of the case seemed to exclude this. He recollected that at that time there had been some ankylosis of the shoulder-joint in the case.

THE PRESIDENT considered the study of the differing resistances in the healthy and diseased arm made by the author interesting, and asked if any member had had experience in such measurements.

Dr. GRAY had noticed much variation in resistance in many of his patients from day to day.

Dr. M. A. STARR, of New York, had measured the resistance in Basedow's disease, and had found it to vary within a thousand ohms in the same cases from time to time. Electricity was diffused through muscular better than through any other tissue. The chief resistance was in the skin. Probably but little of the current permeated nerves, and hence alterations of nervous tissue would not have much to do with the variations mentioned by the author.

Dr. G. M. HAMMOND, of New York, stated that it was well known that the resistance differed from day to day in animals. He asked if in this case it had been measured for a number of times, and was answered that eight trials had been made.

Dr. BIRDSALL thought the question of resistance had little to do with our study of disease. It was easy to explain the numerous variations by the wide differences in vascularity and moisture of the tissues at different times. It might be due in this case to paralysis affecting the physical condition of the tissues. The saturation of the epidermis by perspiration would explain the variations mentioned by Dr. Gray. He had made measurements in cases of Basedow's disease some time ago, and believed that the diminished resistance found was due to the moisture of the skin. It could not depend on dynamic conditions in the nervous system, but was purely a matter of physics about which there was no great mystery.

Dr. GRAY said the explanation by moisture of the skin was not applicable in his cases. The differences which he had observed had not been owing to the humidity of the atmosphere, although atmospheric conditions might possibly induce dynamic changes in the body.

Dr. SACHS pointed out that on one day the author's patient showed 580 ohms increase on the diseased side, and on another day 1,170 ohms increase on the sound side. Such variations evidently had little to do with the pathological process in the patient. Eulenburg had measured the resistances in cases of paralysis agitans and of Basedow's disease, but without any practical results. As such measurements were very complex, they required exceedingly great care.

Dr. PRINCE objected to the use of the palm of the hand for precise experimentation, owing to the great variability of the thickness of the skin, and consequently of the resistance. He thought the forearm ought to be used.

Dr. LESZYNSKY had not brought forward the question of resistance in this case as a diagnostic symptom. It was ncreased in the affected arm at every examination. 'With

the subsidence of the symptoms the resistance gradually diminished, but there was a difference between the sound and morbid sides throughout the disease. As to the question of moisture, there was excessive perspiration upon the paralyzed arm, while the normal arm was dry, in spite of which the resistance was greater upon the former. He thought it better to place the electrode upon the wrist than in the hand. There was no antecedent traumatism in the case; the roughening of the shoulder-joint was the result of the paralysis.

Dr. M. ALLEN STARR, of New York, then presented a specimen of an

INTERPEDUNCULAR MYXOSARCOMA.

It lay in the middle cranial fossa in the median line between the crura cerebri, which it separated. It extended into the lateral ventricles, separating widely the caudate nuclei and optic thalami. The patient was a male, aged twenty-one months at death. At the age of thirteen months, in October last, a lateral nystagmus had been observed in both eyes, varying from time to time. Drs. Knapp and Roosa found a slight pallor of the optic disks, which they considered normal. Later, exophthalmos developed, gradually increasing until death. Convergence of the eyes was impossible, although no paralysis of a cranial nerve was discoverable. The reflexes of the iris were lost. Toward the middle of April, this year, the child became unable to walk. The knee-jerks were exaggerated, there was ankle clonus, and typical spastic rigidity. Finally, the back could not be held up, and later the head could not be supported. There was gradual emaciation. There was no apparent headache. From time to time the scalp was congested. About this time the pallor of the optic disks indicated atrophy. There was no blindness, no hemiopia, no aphasia, as far as could be ascertained in so young a child. Toward the middle of May ataxia of the arms developed, but without loss of muscular power. June 8th, vomiting and Cheyne-Stokes'

respiration came on, and the child died in nine hours. The diagnosis of tumor had been made, but the question of localization was of great interest. Nystagmus was not a localizable symptom, but had been found most frequently in lesions of the corpora quadrigemina. The exophthalmos indicated intracranial pressure; the gait disturbance led one to think of a cerebellar lesion; while the ataxia of the hands pointed to a basilar trouble affecting symmetrically either the pons or the medulla. The question of operative interference had been considered, but he had opposed that proposition because of the difficulty of localizing the tumor. The autopsy proved that the pons was not even pressed upon, and that the cerebellum was normal. There were twenty ounces of fluid in the ventricles. He would ask if there was any localizing value in nystagmus or exophthalmos, and what was the probable cause of the ataxia.

THE PRESIDENT, in referring to the question of ataxia, recalled a case of remarkable bilateral ataxia, with optic atrophy, where a large interpeduncular tumor was found between the crura. He thought such ataxia was to be accounted for by pressure upon the motor tracts or motor nuclei. To him the most puzzling feature of Dr. Starr's case was the absence of blindness. As to the nystagmus, it had as yet, in his opinion, no localizing value. He had seen two cases with lesions of the quadrigeminal bodies, but without nystagmus.

Dr. LESZYNSKY said the child may have had only central vision, which might explain the presence of nystagmus; but Dr. Starr answered that the visional fields were normal.

Dr. SACHS was reminded of Meynert's case of tumor in both optic thalami, with ataxia similar to that of Dr. Starr's case. He thought the thalami might have been pressed upon in the latter, but still was not sure that that would cause the ataxia. He considered nystagnus very frequent in many central disorders of children.

Dr. H. M. LYMAN referred to a case he had seen recently of defective cerebral development in a child where there was also nystagmus.

Dr. GRAY asked how a diagnosis of intracranial tumor had been made so early in this case, and was answered that the diagnosis had not been made until all the symptoms described had made their appearance.

Dr. HAMMOND thought ataxia depended upon injury to the optic thalami or corpora striata, and referred to a case of Weir Michell's in which there was a remarkable unilateral ataxia, with a lesion of the optic thalamus and corpus striatum upon the opposite side.

Dr. LESZYNSKY saw a child, several years ago, with well-marked nystagmus, which disappeared in the course of time. There was no discoverable cause.

The following gentlemen were then elected to active membership: Dr. C. Eugene Riggs, of St. Paul, Minn.; Dr. H. S. Upson, of Cleveland, O.

Dr. B. SACHS, of New York, presented a paper on the

PERONEAL FORM OF PROGRESSIVE MUSCULAR ATROPHY.

The author reiterated his statement of last year that this form was closely related to Duchenne's type. He gave very full details of the cases of two brothers that had recently come under his observation through the kindness of Dr. Gibney. The boys were thirteen and ten years of age. There was a gradual development of double clubfoot in both at the age of five years, followed by an atrophy proceeding upward, beginning in the leg and toe muscles and spreading to those of the thigh; in one case also involving muscles of the upper extremities. The knee-jerks were present. In one there was general anæsthesia, in the other paræsthesia. There was full degenerative reaction in some of the muscles in one boy, partial in the other. The progressive wasting rendered treatment of this form of club-foot less satisfactory than that of congenital cases. He would suspect this peroneal form in all cases where acquired club-foot was associated with progressive wasting of the leg muscles, and par-

ticularly if heredity or family occurrence of the disease could also be established.

Dr. SINKLER could recall several similar cases; one, in particular, of two brothers. But doubtless more would be seen if careful attention were paid to the matter.

Dr. BIRDSALL did not think the presence of knee-jerk so important a diagnostic point as the author seemed to regard it. He had seen a few cases of old poliomyelitis where the paralysis and atrophy were below the knee, and yet the knee-jerks were quite active on both sides. It could not therefore be an essential point in diagosis.

Dr. BULLARD had also observed the presence of the knee-jerk in old cases of poliomyelitis.

Dr. PRINCE asked if the author had said that the absence of pseudo-hypertropy was a diagnostic point between this form and primary myopathies, and was answered in the affirmative. He did not consider this true.

Dr. SPITZKA asked if the symmetry and the coincidence of time and intensity as shown in the photographs were always the case, and was answered in the affirmative.

Dr. SINKLER corroborated two of the speakers as to the presence of the knee-jerk in cases of old poliomyelitis, and cited an instance from his own experience.

Dr. GRAY objected to the division of progressive muscular atrophy into groups. Why should there be an arm type, a face type, a leg type? Such division might be carried out indefinitely. A more useful classification would be upon the pathology of the disease—a division into central, muscular, and peripheral nerve-lesions.

Dr. SACHS said he did not lay great stress upon the presence or absence of the knee-jerk. Yet in extreme atrophy of the vasti from poliomyelitis the knee-jerk was always absent; and if it were present, he should consider it a case of progressive muscular atrophy. He had himself tried to discard subdivision as much as possible, but the present classification was a clinical necessity. A better might be made when the pathology is more accurately determined. At present there was spinal and non-spinal cases, but there was no certainty as regarded peripheral nerve cases.

Dr. MORTON PRINCE, of Boston, then exhibited some microscopic specimens from the muscles of a case of

PSEUDO-HYPERTROPHY.

The patient was now twenty-eight years of age. The specimens showed a large quantity of connective tissue, hypertrophy of a few fibres, and great atrophy of many of the fibres. There was also great loss of striation, but no fatty or granular degenerations, and no vacuolization.